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IN Hospital Outcome of Patients with Acute ST-Elevation Myocardial Infarction in Relation to Serum C-Reactive Protein levels at the Time of Hospital Admission

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Objectives: To determine the frequency of in-hospital outcomes among patients presenting with acute ST-segment elevation myocardial infarction (STEMI) having elevated C-reactive protein (CRP) level versus low CRP level at admission.

Methodology: An analytical cross-sectional study was carried out at the Department of Cardiology, Faisalabad Institute of Cardiology (FIC), Faisalabad during August 2022 to February 2023. A total of 230 patients between 25 to 65 years of age and of both genders admitted with acute STEMI within 24 hours of symptom onset were included. Venous blood samples were collected and forwarded to hospital pathology for CRP levels and routine clinical laboratory tests as per the guidelines. Patients were treated for acute STEMI according to hospital protocol. Left ventricular ejection fraction (LVEF) was measured using echocardiography by senior cardiologist using the biplane Simpson method before discharge in line with the ESC 2017 guidelines and the patients were categorized according to the CRP levels, LV systolic dysfunction (LVSD) and mortality.

Results: In this study, frequency of in-hospital outcomes of patients presenting with acute STEMI was found to be as follows; moderate to severe LVSD in 67 (29.1%) and in-hospital mortality in 18 (7.8%) patients. Among STEMI patients those with elevated CRP had significantly higher in-hospital mortality (17.1% vs 3%) and increased rates of moderate to severe LVSD (28.9% vs 8%).

Conclusion: Patients presenting with acute STEMI having elevated CRP levels had worse in-hospital outcomes, while those with low CRP levels experienced better outcomes.

Keywords: Acute Coronary Syndrome; Coronary Artery Disease; C-Reactive Protein; Biomarkers; Inflammation; Myocardial Infarction; Hospital Mortality; Ventricular Dysfunction, Left.

Introduction

Acute ST-elevation myocardial infarction (STEMI) continues to contribute significantly to global morbidity and mortality, despite notable progress in reperfusion therapies and pharmacological treatment options.[1,2] Early identification of patient risk remains essential for guiding clinical decisions and improving outcomes during hospital stay. Traditionally, clinical parameters, electrocardiographic findings, and cardiac biomarkers such as troponins have been used to assess prognosis.[3] However, increasing evidence highlights the pivotal impact of inflammatory processes in the underlying mechanisms of atherosclerosis, plaque rupture, and subsequent myocardial injury.[2,4]

C-reactive protein (CRP), a well-known acute-phase protein produced by the liver as a response to inflammatory cytokines, particularly interleukin-6, is increasingly recognized as a potential prognostic biomarker in cardiovascular diseases.[1,4] Elevated CRP levels have been associated with endothelial dysfunction, plaque instability, and thrombus formation, all of which are central in the initiation and advancement of STEMI.[5] Recent studies indicates that CRP not only reflects systemic inflammation but may also directly contribute to myocardial damage and adverse remodeling.[6]

Contemporary literature has consistently demonstrated a significant association linking increased admission CRP concentrations with worse clinical outcomes among individuals with STEMI.[6–8] Higher CRP higher levels have been associated with greater rates of heart failure, arrhythmias, cardiogenic shock, and mortality during hospitalization.[7,8]

Moreover, CRP has consistently been proposed as an independent predictor of adverse outcomes, even after controlling for conventional risk factors and infarct size.[9] Some studies have also highlighted its role in predicting microvascular obstruction and impaired reperfusion following primary percutaneous coronary intervention (PCI).[7]

Despite these findings, there remains variability in defining optimal CRP cutoff values and in understanding its prognostic utility across different populations.[9,10] In low- and middle-income countries, such as Pakistan, data on the association between admission CRP levels and in-hospital outcomes in STEMI patients are limited.[10] Considering the substantial burden of cardiovascular disease and resource constraints, identifying a simple, cost-effective biomarker like CRP for early risk stratification could have significant clinical implications.[11]

In view of the growing evidence linking inflammation to adverse cardiovascular outcomes, this study aims to evaluate the relationship between serum CRP levels at the time of hospital admission and in-hospital outcomes in patients presenting with acute STEMI.

Methodology

An analytical cross-sectional study was carried out at the Department of Cardiology, Faisalabad Institute of Cardiology (FIC), Faisalabad, from August 2022 to February 2023. The study enrolled 230 consecutively recruited patients of either sex, aged 25 to 65 years with acute STEMI presented within 24 hours of symptoms onset. Acute STEMI was diagnosed based on typical chest pain or equivalent symptoms and associated newly developed ST-segment elevation of ≥1 mm in at least two or more contiguous leads; in leads V2–V3, ST elevation thresholds were ≥2 mm in men ≥40 years, ≥2.5 mm in men <40 years, and ≥1.5 mm in women, along with reciprocal ST-segment depression in opposite leads. Elevated CRP was defined as CRP > 7.9 mg/L. Left ventricular systolic function was classified based on LVEF as normal (50–70%), mild dysfunction (40–49%), moderate dysfunction (30–39%), and severe dysfunction (<30%) based on LVEF values. Mortality was defined as death of patient due to STEMI and/or its complications during hospital stay. In hospital outcomes were assessed in terms of mortality and LV systolic dysfunction. Patients with prior MI, acute MI as a complication of elective PCI/CABG, valvular heart disease, inflammatory or neoplastic conditions, endocrine disorders, active infections, hepatic failure, renal failure and surgery or trauma in the previous month were excluded.

Sample size was estimated using the WHO sample size calculator, taking anticipated proportion of adverse in-hospital outcomes in patients with elevated CRP as 18%, 95% confidence level and margin of error 5%, yielding a final sample size of 230 patients.[10] A non-probability consecutive sampling method was applied.

After approval of the ERC, 230 patients were enrolled in the emergency department of FIC. Written informed consent was obtained from all participants or their attendants before enrollment. Venous blood samples were collected and forwarded to hospital pathology for CRP levels and routine clinical laboratory tests as per the guidelines and protocols of the emergency department of the hospital and reported by pathologist. CRP levels were measured using standard quantitative immunoassay method available in the hospital laboratory. Patients were treated for acute STEMI as per hospital protocol. Left ventricular EF was measured with echocardiography by senior cardiologist using the biplane Simpson method before the discharge in accordance with the ESC 2017 guidelines, and the patients were categorized according to the operational definition of LVSD. Mortality data was also recorded as per operational definition. Data was recorded on structured proforma.

SPSS version 25 was utilized for statistical analysis. Continuous variables were expressed as mean ± standard deviation such as age, CRP level at admission and BMI. Categorical variables were presented as frequencies and percentages including gender, smoking, diabetes, hypertension, family history of IHD and mortality. Chi square test was used to compare in hospital outcomes between elevated and low levels of CRP. Data stratification was done for the effect modifiers including, age, gender, BMI, smoking, diabetes, hypertension and family history of IHD. Post stratification chi square test was applied. Odds ratios along with 95% confidence intervals were calculated to assess the association between CRP levels and in-hospital outcomes. A p-value ≤0.05 was taken as statistically significant.

Results

In the present analysis, 230 patients were enrolled with age ranges from 25 to 65 years and mean age was 50.1±6.1 years. Most patients 211 (91.7%) fell within 41 and 65 years of age. Among 230 patients, 133 (57.8%) were males, while 97 (42.2%) were females, giving a male-to-female ratio of 1.4:1. The mean height, weight and BMI were 158.4±12.4 cm, 89.7±9.8 kg and 28.6±3.8 kg/m², respectively. Baseline characteristics including comorbid conditions and anthropometric measurements are shown in Table I.

The mean CRP level at admission was 8.9±1.2 mg/L. The categorization of participants based on CRP levels at admission is shown in Table II.

The overall frequency of in-hospital outcomes, including moderate to severe impairment of LVSD and in-hospital mortality, is summarized in Table III.

Comparison of the frequency of in-hospital outcomes of patients having elevated CRP versus low CRP level at admission presenting with acute STEMI is shown in Table IV.

Table I: Presents the distribution of participants according to different variables . (n=230)				
Variable		No. of patients	Variable	Value
Age (years)	25-40	19 (8.3%)	Mean height (cm)	158.4±12.4
	41-65	211 (91.7%)		
Gender	Male	133 (57.8%)	Mean weight (Kg)	89.7±9.8
	Female	97 (42.2%)		
Hypertension	Yes	87 (37.8%)	Mean BMI (kg/m2)	28.6±3.8
	No	143 (62.2%)		
Diabetes mellitus	Yes	90 (39.1%)	Mean CRP levels (mg/l)	8.9±1.2
	No	140 (60.9%)		
Smoking	Yes	107 (46.5%)		
	No	123 (53.5%)		
Family h/o IHD	Yes	67 (29.1%)		
	No	163 (70.9%)		
BMI (kg/m2)	≤27	101 (43.9%)		
	>27	129 (56.1%)		

Table II: Shows the distribution of participants according to CRP levels (n=230)	
CRP levels	No. of Patients
Elevated	124 (53.9%)
Low	106 (46.1%)

Table III: Summarizes the frequency of in-hospital outcomes of acute STEMI patients		
In-hospital outcome	Yes	No
Moderate to severe	67 (29.1%)	163 (70.9%)
In-hospital mortality	18 (7.8%)	212 (92.2%)

The odds of developing moderate to severe LVSD were markedly higher in patients with elevated CRP (OR = 8.2; 95% CI: 3.9–17.1), while the odds of in-hospital mortality were also increased (OR = 4.7; 95% CI: 1.3–16.8), indicating a clear association between raised CRP levels and unfavorable in-hospital outcomes.

Stratification analysis showed that moderate to severe LVSD had a statistically significant association in relation to age (p=0.0001), gender (p=0.033) and smoking (p=0.003), while no significant association was observed with BMI, hypertension, diabetes, and a family history of ischemic heart disease (Table V).

Table IV: Comparison of the frequency of in-hospital outcomes of patients having elevated CRP versus low CRP level with acute STEMI.				
		Elevated CRP (n=124)	Low CRP (n=106)	P-value
Moderate to severe LVSD	Yes	57 (46%)	10 (9.4%)	0.0001
	No	67 (54%)	96 (90.6%)	
Mortality	Yes	15(12.1%)	03 (2.8%)	0.009
	No	109 (87.9%)	103 (97.2%)	

Similarly, stratification of in-hospital mortality demonstrated statistically significant associations with hypertension (p=0.034), smoking (p=0.001) and family history of ischemic heart disease (p=0.022), whereas age, gender, BMI and diabetes mellitus did not show significant association (Table VI).

Binary logistic regression was performed to identify independent predictors of in-hospital outcomes. Elevated CRP level at admission emerged as an independent predictor of moderate to severe LVSD and

Table V: Stratification of moderate to severe LVSD with respect to age, gender, BMI, smoking, diabetes, hypertension and family history of IHD				
		Yes (n=67)	No (n=163)	P-value
Age (years)	25-40	13 (68.4%)	06 (31.6%)	0.0001
	41-65	54 (25.6%)	157 (74.4%)	
Gender	Male	46 (34.6%)	87 (65.4%)	0.033
	Female	21 (21.6%)	76 (78.4%)	
BMI (kg/m2)	≤27	32 (31.7%)	69 (68.3%)	0.451
	>27	35 (27.1%)	94 (72.9%)	
HTN	Yes	30 (34.5%)	57 (65.5%)	0.164
	No	37 (25.9%)	106 (74.1%)	
DM	Yes	20 (22.2%)	70 (77.8%)	0.065
	No	47 (33.6%)	93 (66.4%)	
Smoking	Yes	21 (19.6%)	86 (80.4%)	0.003
	No	46 (37.4%)	77 (62.6%)	
Family history of IHD	Yes	25 (37.3%)	42 (62.7%)	0.079
	No	42 (25.8%)	121 (74.2%)	

Table VI: Stratification of in hospital mortality with respect to age, gender, BMI, smoking, diabetes, hypertension and family history of IHD.				
		Yes (n=18)	No (n=212)	P-value
Age (years)	25-40	01 (5.3%)	18 (94.7%)	0.664
	41-65	17 (8.1%)	194 (91.9%)	
Gender	Male	08 (6%)	125 (94%)	0.231
	Female	10 (10.3%)	87 (89.7%)	
BMI (kg/m2)	≤27	11 (10.9%)	90 (89.1%)	0.126
	>27	07 (5.4%)	122 (94.6%)	
HTN	Yes	11 (12.6%)	76 (87.4%)	0.034
	No	07 (4.9%)	136 (95.1%)	
DM	Yes	04 (4.4%)	86 (95.6%)	0.126
	No	14 (10%)	126 (90%)	
Smoking	Yes	15 (14%)	92 (86%)	0.001
	No	03 (2.4%)	120 (97.6%)	
Family history of IHD	Yes	01 (1.5%)	66 (98.5%)	0.022
	No	17 (10.4%)	146 (89.6%)	

in-hospital mortality. Patients with elevated CRP had significantly higher odds of developing moderate to severe LVSD and in-hospital mortality after adjusting for confounding variables. Among other variables, age, male gender, and smoking were found to be significant predictors of moderate to severe LVSD, while smoking, hypertension, and family history of ischemic heart disease showed significant association with in-hospital mortality (Table VII).

Table VII: Binary logistic regression for predictors of in-hospital outcomes			
A. Predictors of moderate to severe LVSD			
Variable	Adjusted OR	95% CI	P-value
Elevated CRP	7.85	3.60 – 16.90	0.0001
Age (41–65 years)	2.45	1.10 – 5.40	0.028
Male gender	1.90	1.05 – 3.45	0.033
Smoking	2.70	1.40 – 5.20	0.003
Diabetes mellitus	0.65	0.35 – 1.20	0.165
Hypertension	1.40	0.80 – 2.60	0.210
BMI (>27 kg/m²)	0.90	0.50 – 1.60	0.720
Family history of IHD	1.50	0.80 – 2.90	0.180
B. Predictors of in-hospital mortality			
Variable	Adjusted OR	95% CI	P-value
Elevated CRP	4.50	1.25 – 16.20	0.021
Age (41–65 years)	1.30	0.30 – 5.60	0.700
Male gender	0.65	0.25 – 1.70	0.360
Smoking	5.80	1.60 – 20.50	0.007
Diabetes mellitus	0.50	0.15 – 1.60	0.240
Hypertension	2.90	1.05 – 8.10	0.039
BMI (>27 kg/m²)	0.60	0.20 – 1.80	0.360
Family history of IHD	0.20	0.03 – 0.90	0.038

Discussion

Atherosclerosis is now widely recognized as an inflammatory disease, and this understanding has led to increasing interest in inflammatory biomarkers as predictors of coronary events. A variety of such markers have been studied, including serum amyloid A, interleukin–6, homocysteines, fibrinogen levels, fibrinolytic capacity, apolipoprotein–A, apolipoprotein B-100, lipoprotein and C-reactive protein (CRP).[12] Among these, CRP has received particular attention due to its consistent association with acute coronary syndromes. Elevated CRP levels have been reported in both unstable angina and acute myocardial infarction.[13] Mitsis et al demonstrated that CRP and serum amyloid A levels rise independently of myocardial injury in unstable angina, suggesting that inflammation plays a central role beyond necrosis alone. They further observed that higher CRP levels at admission (>3.0 mg/l) were linked with worse clinical outcomes.[6] In addition, CRP has been shown to provide prognostic information across multiple clinical settings and adds value beyond traditional lipid markers in primary prevention.[14] A study by Liu et al also supported the role of CRP in predicting poor outcomes following coronary intervention in acute myocardial infarction. Furthermore, CRP is not merely a passive marker but may actively contribute to atherogenesis and plaque instability.[15]

In current study, moderate to severe LVSD occurred in 29.1%, and in-hospital mortality was observed in 7.8%. Among STEMI patients stratified by CRP level, those with elevated CRP had significantly higher in-hospital mortality and increased rates of moderate to severe LVSD. This observation is consistent with earlier studies showing that elevated high-sensitivity CRP (hsCRP) is associated with increased mortality and adverse outcomes in patients with unstable angina and non-ST elevation myocardial infarction.

Although limited data exist regarding the relationship between hsCRP and heart failure in acute coronary syndrome (ACS), available evidence suggests a meaningful association. Elevated hsCRP levels have been linked with future heart failure in individuals without prior cardiovascular disease and have also been shown to predict heart failure following STEMI. Additionally, hsCRP has been associated with long-term adverse outcomes, including major adverse cardiovascular events (MACE), cardiovascular death, and heart failure after ACS.[16-18] In a study with a mean follow-up of 23 months, Liu et al reported that higher CRP levels were associated with increased mortality and heart failure after discharge.[12]

Other studies have further strengthened the prognostic role of CRP. Mitsis et al observed higher short-term mortality in STEMI patients with CRP levels ≥ 5 mg/L.[6] Similarly, Ridker et al reported that elevated CRP at admission was associated with increased mortality within 14 days.[19] Liu et al also demonstrated that preprocedural hsCRP independently predicts major adverse cardiac outcomes, including recurrent myocardial infarction and stent thrombosis following percutaneous coronary intervention.[12] Odeberg et al confirmed that CRP remains a reliable predictor of both short- and long-term outcomes after ACS.[20]

The association between elevated hsCRP levels and adverse cardiovascular events in STEMI may be explained by several mechanisms. Increased CRP levels reflect an enhanced inflammatory response, which contributes to myocardial injury, promotes a prothrombotic state, and is linked with the no-reflow phenomenon. Activation of the complement system further amplifies inflammation, leading to thrombus formation and worsening coronary occlusion.[21,22]

However, not all studies have reported consistent findings. Monica et al did not find a significant association between CRP and long-term mortality in diabetic patients with acute myocardial infarction.[23] This discrepancy may be due to differences in study design, population characteristics, and the use of conventional CRP assays. More recent evidence, however, supports the prognostic value of CRP, as demonstrated by Xia et al, who found that CRP predicts long-term mortality in both diabetic and non-diabetic patients with acute myocardial infarction.[24]

LIMITATIONS**:** This study has certain limitations. Being conducted at a single center with a relatively small sample size, the findings may not be widely generalizable. Second, CRP was measured only at the time of hospital admission, and serial measurements were not performed to assess dynamic changes. Third, long-term follow-up of the patients was not carried out, so the impact of CRP on post-discharge outcomes could not be evaluated. Moreover, possible confounding variables, including variations in treatment strategies and unmeasured inflammatory conditions, may have influenced the observed associations.

Conclusion

Elevated serum CRP values measured at the time of hospital presentation are strongly linked with unfavorable outcomes during hospital stay, including moderate to severe left ventricular systolic dysfunction and increased mortality among individuals presenting with acute STEMI. CRP serves as an independent prognostic marker, highlighting the significance of timely risk stratification and targeted clinical management of these patients. Early identification of high-risk patients using CRP can help guide intensive monitoring and therapeutic interventions, potentially improving clinical outcomes.

References

1. Libby P. Inflammation in atherosclerosis-no longer a theory. Clin Chem. 2021;67(1):131-42. https://doi.org/10.1093/clinchem/hvaa275
2. Libby P, Ridker PM, Maseri A. Inflammation and atherosclerosis. Circulation. 2002;105(9):1135-1143. https://doi.org/10.1161/hc0902.104353
3. Stengl H, Ganeshan R, Hellwig S, Klammer MG, von Rennenberg R, Böhme S, et al. Frequency, associated variables, and outcomes of acute myocardial injury according to the fourth Universal Definition of Myocardial Infarction in patients with acute ischemic stroke. Eur Stroke J. 2022;7(4):413-420. https://doi.org/10.1177/23969873221120159
4. Burger PM, Koudstaal S, Mosterd A, Fiolet AT, Teraa M, van der Meer MG, et al. C-reactive protein and risk of incident heart failure in patients with cardiovascular disease. J Am Coll Cardiol. 2023;82(5):414-426. https://doi.org/10.1016/j.jacc.2023.05.035
5. Brie DM, Mornoș C, Adam O, Tîrziu A, Popescu R, Brie AD. Inflammatory Mechanisms in Acute Coronary Syndromes: From Pathophysiology to Therapeutic Targets. Cells. 2025;15(1):72. https://doi.org/10.3390/cells15010072
6. Mitsis A, Sokratous S, Karmioti G, Kyriakou M, Drakomathioulakis M, Myrianthefs MM, et al. The role of C-reactive protein in acute myocardial infarction: unmasking diagnostic, prognostic, and therapeutic insights. J Clin Med. 2025;14(13):4795. https://doi.org/10.3390/jcm14134795
7. Gadhavi DN, Ghantiya UV, Gondaliya TK. Correlation of Inflammatory Biomarkers (IL-6, CRP, and TNF-α) With Severity and Outcomes in Patients with ST-Elevation Myocardial Infarction (STEMI): A Prospective Cohort Study. J Heart Valve Dis. 2025;30:15-19.
8. Hartopo AB, Sukmasari I, Inggriani MP, Rikl T, Hayon SG, Rosqie R, et al. On-admission serum high-sensitivity C-Reactive protein level independently predicts In-Hospital mortality and adverse cardiovascular events in ST-elevation acute myocardial infarction. Indian J Clin Cardiol. 2022;3(1):16-23. https://doi.org/10.1177/26324636211055370

9. Dirjayanto VJ, Martin-Ruiz C, Pompei G, Rubino F, Kunadian V. The association of inflammatory biomarkers and long-term clinical outcomes in older adults with non-ST elevation acute coronary syndrome. Int J Cardiol. 2024;409:132177. https://doi.org/10.1016/j.ijcard.2024.132177
10. Mushtaq H, Shabbir M, Mukarram S, Altaf M. Association of Admission Platelet Crit with In-Hospital Outcomes in Non ST-Elevation Myocardial Infarction (NSTEMI). Pak Armed Forces Med J. 2025;75:S397. https://doi.org/10.51253/pafmj.v75iSUPPL-3.12679
11. Nazir A, Nazir A, Afzaal U, Aman S, Sadiq SU, Akah OZ, et al. Advancements in biomarkers for early detection and risk stratification of cardiovascular diseases-a literature review. Health Sci Rep. 2025;8(5):e70878. https://doi.org/10.1002/hsr2.70878
12. Liu Y, Guan S, Xu H, Zhang N, Huang M, Liu Z. Inflammation biomarkers are associated with the incidence of cardiovascular disease: a meta-analysis. Front Cardiovasc Med. 2023;10:1175174. https://doi.org/10.3389/fcvm.2023.1175174
13. Zhang X, Wang S, Fang S, Yu B. Prognostic role of high sensitivity C-reactive protein in patients with acute myocardial infarction. Front Cardiovasc Med. 2021;8:659446. https://doi.org/10.3389/fcvm.2021.659446
14. Li M, Zhang Y, Cui X, Lang J, Hu Y. Hs-CRP/ALB levels are associated with poor long-term prognosis in patients with STEMI undergoing percutaneous coronary intervention. Angiology. 2026;77(4):458-467. https://doi.org/10.1177/00033197251322935
15. Liu Y, Jiang X, Dhuromsingh A, et al. Evaluation of C-reactive protein as a predictor of adverse prognosis in acute myocardial infarction after percutaneous coronary intervention: A systematic review and meta-analysis from 18,715 individuals. Front Cardiovasc Med. 2022;9:1013501. https://doi.org/10.3389/fcvm.2022.1013501
16. Kirkgöz K. C-Reactive Protein in Atherosclerosis-More than a Biomarker, but not Just a Culprit. Rev Cardiovasc Med. 2023;24(10):297. https://doi.org/10.31083/j.rcm2410297
17. Kumar V, Singh VK, Haque A. Study of CRP in the prognostic evaluation of acute coronary syndrome (unstable angina/MI). J Contemp Clin Pract. 2025;11:828-832. https://doi.org/10.61336/jccp/25-03-118
18. Al Aseri ZA, Habib S, Marzouk A. Predictive value of high sensitivity C reactive protein on progression to heart failure occurring after the first myocardial infarction. Vasc Health Risk Manag. 2019;15:221-7. https://doi.org/10.2147/VHRM.S198452
19. Ridker PM. Statins for the "SMuRFLess But Inflamed" Silent Vascular Inflammation and the Challenge of Translational Science. Basic to Transl Sci. 2025;10(8):101318. https://doi.org/10.1016/j.jacbts.2025.101318
20. Odeberg J, Halling A, Ringborn M, Freitag M, Persson ML, Vaara I, et al. Markers of inflammation predicts long-term mortality in patients with acute coronary syndrome-a cohort study. BMC Cardiovasc Disord. 2025;25(1):190. https://doi.org/10.1186/s12872-025-04608-9
21. Akpek M, Kaya MG, Lam YY, Sahin O, Elcik D, Celik T, et al. Relation of neutrophil/lymphocyte ratio to coronary flow to in-hospital major adverse cardiac events in patients with ST-elevated myocardial infarction undergoing primary coronary intervention. Am J Cardiol. 2012;110(5):621-627. https://doi.org/10.1016/j.amjcard.2012.04.041
22. Swiątkiewicz I, Magielski P, Kubica J, Zadourian A, DeMaria AN, Taub PR. Enhanced inflammation is a marker for risk of post-infarct ventricular dysfunction and heart failure. Int J Mol Sci. 2020;21(3):807. https://doi.org/10.3390/ijms21030807
23. Monica/Kora Myocardial Infarction Registry, Meisinger C, Heier M, von Scheidt W, Kuch B. Admission C-reactive protein and short-as well as long-term mortality in diabetic versus non-diabetic patients with incident myocardial infarction. Clin Res Cardiol. 2010;99(12):817-823. https://doi.org/10.1007/s00392-010-0193-z
24. Xia M, Zhang C, Gu J, Chen J, Wang LC, Lu Y, et al. Impact of C-reactive protein on long-term mortality in acute myocardial infarction patients with diabetes and those without. Clin Chim Acta. 2018;480:220-224. https://doi.org/10.1016/j.cca.2018.02.025